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# Remarks on Pathogenic Bacteria.

### BY DR. H. J. DETMERS, F. R. M. S.

MR. PRESIDENT AND MEMBERS OF THE AMERICAN SOCIETY OF MICROSCOPISTS:—It was my intention to prepare a paper to be read before the Society at this meeting, but circumstances beyond my control prevented its preparation,consequently,when coming here,I came to learn and to listen, and not to speak. But as you, Mr. President, desire that I shall say something, although entirely unprepared, the members of the Society, I hope, will be forbearing, and be satisfied with such a rambling talk, as I shall be able to give. It was my intention to write on "The Actions of Pathogenic Bacteria," (the name *bacteria* used as a generic term), because in my investigations of infectious diseases of live stock, I could not help observing that pathogenic bacteria do not all act alike. So, for instance, while many, perhaps most of the known pathogenic bacteria—particularly *Bacillus anthracis*—act as a ferment, cause fermentation or decomposition of the blood, others, for instance the micrococci, or more correctly, diplococci of swine-plague, do not produce any chemical changes in the blood and other tissues, except such, as will necessarily result from the withdrawal of such matter, as may be appropriated by them for their existence, and for their development and propagation. Otherwise they only act in a mechanical way; they close the finer capillaries, thus cause local congestion and embolism, and in that way produce the morbid process,—their action, at any rate, cannot be called a specific one. That such is the case, I have proved as early as 1878 and 1879, as will be seen by reading my reports to the Commissioner of Agriculture. Nevertheless,M.Pasteur, as late as last year, claims the discovery of the diplococci of swine-plague as his. But so it goes, certain Frenchmen, and M. Pasteur, although otherwise a great man, is one of them, are ever-ready to appropriate any convenient piece of literary or scientific property that is not closely guarded, or not kept under lock and key.

In my investigations I made another observation, that is, the same pathogenic bacteria may, under certain conditions and surroundings, be very malignant, and be producing disease in every instance, while under others they appear to be far less malignant, or even harmless.

An abundance of proof that such is the case can be furnished. In the first place, it is a well-known fact that almost all epidemic and epizootic diseases, but particularly such, as are unquestionably caused by bacteritic growths, are more malignant at one time, or in one year, than at other times or in other years, and cause more losses in one epidemy or epizooty as the case may be, than in another, that many persons or animals respectively, though much exposed to infections, remains exempt, or do not take the disease, although there can be hardly any doubt that while exposed, their organisms have taken up a larger or smaller number of pathogenic bacteria. Such—that in every epidemic some persons, and in every epizootic some animals, remained exempt—at least, was the case in regard to all those infectious diseases that came under my observation, and I am sure, any practicing physician will testify to the same fact. Proof, however, would be more definite, if the blood, etc., of persons or animals thus exposed, but not affected by disease, had been oftener subjected to a microscopic examination.

Some time since Drs. Brown and Curtis, of this city (Chicago), attended a boy affected with tetanus. The boy had been fooling with the toy-pistol. Dr. Curtis examined the boy's blood (taken from a puncture in the finger) and found numerous micrococci. The doctors thought they had made a discovery, and searched for the probable source of those micrococci. Near the house they found a stagnant pool of water, and Dr. Curtis, putting some of the water under the microscope, found in that water, apparently, the same micrococci, at least, as to size and form, as in the boy's blood. The doctors, however, did not stop there in their investigation. Dr. Curtis further examined the blood of the boy's mother and sister, to all appearances perfectly healthy persons, and to his astonishment again found the same micrococci in considerable numbers. Then Dr. Brown concluded the micrococci could not possibly be the cause of tetanus, for, he argued, if they were, the boy's mother and sister must have the same disease. Dr. Curtis did not express an opinion. Of course, I will not, and do not say that micrococci do constitute the cause of tetanus, and all I do say is that the presence of the same micrococci in the blood of two apparently healthy persons, the boy's mother and sister, is no proof whatever that those micrococci, seen in the blood of the boy, did not constitute the cause of the disease. The boy recovered, otherwise Dr. Curtis, in carrying on his research with his usual care and precaution, might have made an important discovery.

If the following facts are taken into consideration, it will not appear to be impossible that the micrococci, after all, bear a causal connection to the disease (tetanus). In the first place, a micrococcus is abundantly small to pass everywhere with the greatest ease where a blood-corpuscle can pass. Secondly, in many infectious diseases, looked upon as being caused by micrococci, the latter may be found in the blood circulating in unaffected parts of the body, but always as micrococci, seldom as diplococci, and *never* as *micrococcus chains* and *zoogloea-masses*, while in the affected tissues the latter forms are abundant. This shows that the formation of zoogloea-masses and chains has something to do with the morbid process. According to my observations, the latter, in swine-plague at least, is the result of embolism, caused by zoogloea-masses becoming lodged in, and thus obstructing, the finer capillaries. As long as a person or an animal is healthy, or as long as the blood is regularly circulating in all parts of the body, no zoogloea-masses, it seems, are formed, and no obstructions occur caused by micrococci, but as soon as the current of blood in some part or parts of the body becomes irregular or retarded, perhaps through a sudden contraction of some capillaries caused by whatever agency it may be—a short exposure to a draft of cold air appears to be sufficient—the conditions, it seems, are given for the formation of zoogloea-masses, and all further consequences follow. Returning to the tetanus case, it is a well-known fact that toy-pistol wounds, and in short all those wounds frequently followed by tetanus, are, as a rule, lacerated, attended with more or less congestion, and, being exceedingly painful, show irritation of the nervous system. The congestion or irregularities in the capillary circulation, it seems, is most pronounced in the capillary system of the irritated nerves. The rest is easily explained, particularly if it is taken into consideration that tetanus in some localities is a frequent, and in others a very rare or entirely unknown disease. If statistics were taken, it would soon be possible to map out the tetanus districts, and it would probably be shown that all those localities in which tetanus is of frequent occurrence, have one thing in common, namely, a high stand of the surface water.

Another illustration is afforded by the Southern, Spanish, or so-called Texas cattle fever, which, undoubtedly, is a bacteritic disease. The pathogenic principle (the bacteria) which seems to develop on the grasses of the South, and is probably taken up by the cattle with drinking stagnant water, or by eating semi-decomposed grasses, does not visibly affect the native cattle, while, on the other hand, cattle imported from the North will take sick, and almost invariably (at least in a majority of cases) will die within a short time, often within a few weeks. But the native southern cattle, if taken from their native range and driven North at the end of the winter, after the spring rains and warmer weather have set in, or, in other words, after the old grass of last year's growth has commenced to decompose, and after new, young grass has made its appearance, will spread death and destruction wherever they go, while they themselves will remain healthy, or at least do not become affected with southern cattle fever. These southern cattle seem to carry and to propogate the pathogenic bacteria of the southern fever in their saliva. At any rate, if northern cattle graze where the grass has been contaminated with the slaver or saliva of such southern cattle, or if they (the northern cattle) drink water from ponds or water-holes, etc., which have been polluted with the slaver of southern cattle, the disease (southern cattle fever) is sure to make its appearance, and to become very destructive among the northern cattle after a period of incubation varying from three to thirteen weeks. But these northern cattle, even if suffering from the fever, are not able to infect other cattle that may come in contact with them, occupy the same pastures, or frequent the same watering places. To put it in a nutshell: cattle that take the disease do not communicate it, either directly or indirectly, and cattle that communicate it do not take it. Further, as already stated, southern cattle, as long as they occupy their native range, possess immunity. But this immunity disappears if the southern cattle are taken a certain distance further south, for instance from Northern Texas or the Indian Territory to Southern Texas, or from Southern Texas to Cuba, or other parts of the West Indies; again, if southern cattle are taken North, and are kept there one winter, because in that case their susceptibility or predisposition will become just the same as that of native northern cattle. These clinical facts, not only observed by myself, but also by a great many others, go far to show that the bacteria of southern cattle fever require, in order to become malignant (pathogenic) certain conditions, which are not existing, or perhaps have become exhausted in the southern cattle while on their native range, but are again reproduced if these same cattle are wintered, even if only one winter, further north, for instance north of the Indian Territory. Also, that further south, for instance, in Cuba, much less of these conditions is required than in Southern Texas, and in Southern Texas much less than in Northern Texas or in the Indian Territory. What these conditions consist in is not, or at least, but partially, known. Difference in climate and temperature seems to be of some influence, but of hardly enough to afford an explanation. Some classes of bacteria, as is well known, after a certain length of time, or after having reached a certain stage of development, exhaust (sterilize) their medium, or the subject they live in, and all propagation ceases till they are transposed to a new, fresh medium. Some species even make a further propagation in the same medium impossible by their own products. So it may happen that in certain infectious diseases, the first attack produces more or less immunity against further attacks. In most bacteritic diseases, however, in swine-plague for instance, this immunity is only partial, and not lasting I have observed that one and the same hog—notwithstanding that a hog under domestication is for obvious reasons only a short-lived animal—suffered three times from swine-plague, and cases in which an animal was attacked a second time are quite numerous. Such cases undoubtedly would be still more frequent if the disease (the first attack) were not so fatal as it generally is. In regard to southern or Texas cattle fever, too, the immunity enjoyed by southern cattle, while on their native range, is invariably destroyed if these cattle are taken north, and kept there over winter. Consequently, M. Pasteur's inoculation theories need not to be tried on so-called Texas cattle fever.

All this, however, hardly affords a sufficient or satisfactory explanation as to the conditions necessary, on the one hand, to the exercise of the malignant properties of pathogenic bacteria, and on the other, to check the action of those malignant properties. I may here remark, I repeatedly found swine-plague micrococci, or, rather, diplococci, in the affected portions of the lungs and in the lymphatic glands of hogs that had recovered, or were recovering, from an attack of swine-plague. I found them four and five weeks after the morbid process had ceased to act, or, perhaps, more correctly, after a retrogressive process had set in. Consequently, those micrococci had become dormant, or had ceased to do mischief, notwithstanding that inoculated into a healthy hog they produced the disease.

We must admit, we have as yet only made a beginning in our knowledge of disease-producing bacteria, and a great deal is yet to be learned and to be explored before the action of pathogenic bacteria, and the conditions necessary to develop, or to stop, respectively, their malignant activity, are fully understood. We have merely gained a peep into a new world, a world the existence of which our fathers had no knowledge of.

A few remarks I wish to make concerning antiseptics, or, rather, an internal use of antiseptic medicines. First, as to carbolic acid. It is a well-known fact that bacteria—pathogenic bacteria and others—are not killed by weak solutions of carbolic acid, or by such solutions as can with safety be given to an animal. But it seems that giving weak solutions of carbolic acid, of iodine, or of several other medicines, usually classed among the antiseptics, creates conditions in the animal organism unfavorable to bacteritic growth, and particularly to any action of the disease-producing properties of the pathogenic bacteria. At any rate, in my numerous (as I have not any of my notes at hand, I cannot state the exact number, which, however, may be learned from my reports to the Commissioner of Agriculture) experiments concerning swine-plague, I invariably found that an inoculation with swine-plague material—fresh lung exudations—failed to produce the disease, if the pig, as soon as inoculated, received every day for two weeks, or during the time which the period of incubation might have lasted, if the inoculation had been effective, a dose of carbolic acid in its water for drinking, amounting to ten drops of 95 per cent. carbolic acid for every hundred pounds of live weight. With small doses of iodine—iodide of potassium and iodine dissolved in water—I had the same results, but the iodine experiments were not continued, because the iodine solution proved to have otherwise a very unfavorable effect upon the animal organism. Some other antiseptics were tried, but the same proved either to be too expensive, or not sufficiently reliable to be of any practical value. Although the antiseptics (carbolic acid, iodine, etc.), administered in the water for drinking, invariably prevented a development of the morbid process, in some (few) cases the inoculation was followed by a slight reaction, consisting in a slight rise of temperature, sometimes preceded by a slight chill, and attended with more or less diminution of that natural vivacity peculiar to a perfectly healthy pig. In all those cases, however, in which the carbolic acid treatment was not commenced until the morbid process had fairly developed, or until important morbid changes had been produced, the antiseptic, beyond perhaps somewhat retarding the morbid process, had but little or no effect—did not save the animal's life. That in the other cases, in which the carbolic acid, iodine, etc., treatment was begun immediately after the inoculation had been performed, the antiseptics were effective in preventing the outbreak of the disease, there can be no doubt, because in every one of my numerous inoculation experiments (see reports to the Commissioners of Agriculture), in which no antiseptics were used, every inoculation of a healthy pig with fresh lung exudation was invariably and in due time (usually in five to seven days) followed by a development of the very characteristic morbid process, notwithstanding that in all cases the inoculation was made in the ear with a small inoculation needle, with which not more than about one-fourth of a drop of fresh lung exudation was inserted. I may yet state, in all my inoculation experiments I endeavored to use none but healthy pigs, and undiluted virus, or material direct from a diseased animal was inserted. I never used anything but perfectly fresh lung exudation. An inoculation with putrid lung exudation, or lung exudation swarming with *Bacterium termo*, may cause septicæmia, but will not produce swine-plague. Now, in what way, or by what changes produced, did carbolic acid prevent the development of the morbid process, or check the morbific action of the swine-plague bacteria? In the dilution given, it could not, and did not, directly kill them. If it had killed them the slight reaction which, in a few cases, followed the inoculation, would have been hardly possible. Consequently, it must have produced changes, or created conditions, not suitable to the exercise of the malignant (pathogenic) properties of the bacteria. It, perhaps, prevented the formation of zoogloea-masses, something upon which the whole pathogenic power of the swine-plague bacteria seems to rest. A swine-plague micrococcus or diplococcus is abundantly small to pass with the greatest facility wherever a blood-disk is able to pass, consequently cannot cause any stagnation in the capillaries, but as soon as zoogloea-masses, often a great deal larger than a blood-disk, are formed, stagnation or partial stagnation in some of the finer capillaries, embolism, and in consequence exudation, and often extravasations of blood, processes which constitute the basis of all the morbid changes, are inevitable. This also explains why full-grown, vigorous animals, with firmer capillaries, and a strong heart-muscle, older boars for instance, often recover, where every younger pig is bound to die. Still, in what way the carbolic acid is acting, besides materially reducing the temperature of the animal, I will not attempt to explain.

To return to the southern cattle fever. I am sorry to say, an opportunity to make more extensive observations, and to verify by experiments those observations I have been able to make, has not been given me. I am working under instructions, and those instructions have to be obeyed. One thing, though, I may yet mention, that is, I succeeded in several cases in checking the progress of the morbid process of southern cattle fever, which mainly develops in the liver, and (secondarily) in the spleen,by giving extraordinarily large doses of chinoidin (amorph. quinin), doses amounting to one drachm for every hundred pounds of live weight. A very marked reduction of temperature, about four degrees within twelve hours, regularly followed, and the disease abated.

In conclusion, Mr. President, allow me to say a word in regard to the classification of bacteria, schizophytes, or schizomycetes. As remarked before, we just stand at the threshold of what may be learned in regard to these minute and simple organisms, which will probably be considered in the future as a fourth kingdom, already named by Haekel, " Protista." Consequently our present classifications cannot be expected to be perfect. That of Cohn probably is the best, but it, too, can not lay any claim to perfection. So, for instance, it seems to me, the genus micrococcus has no real foundation, because according to my observations a micrococcus is not a perfect or mature bacterium (bacterium used as a generic term), but only a transitory or unripe form. Micrococci, wherever the conditions are favorable, soon form zoogloea-masses. In these zoogloea-masses the single cocci soon become double, form diplococci, then the glia breaks, whether on account of a consumption or absorption of the viscous mass, which constitutes the glia, by the cocci, or whether on account of the increased bulk of its contents caused by the growth of the cocci, that is, their change into diplococci, I do not know. The diplococci and the yet unchanged micrococci become free or gradually separated from the glia, but the development or propagation does not cease: on the contrary, proceeds with increased rapidity. The single cocci (micrococci), or most of them, soon become double, and every diplococcus in a short time doubles itself at both ends, and forms a small chain, first of 4, then of 8, then of 16, etc., links; then the chains, when grown to some length, commence to separate, or to break up into shorter chains, or even into diplococci, but *never* in smaller portions than a double or figure ∞ micrococcus (diplococcus). At least I never saw a single micrococcus separate itself from a chain, or from a diplococcus, consequently the single micrococci must have a different source, and must proceed from so-called lasting spores, *which somtimes can be found developed among some diplococci that have become separated from a diplococcus chain.* Consequently, I have come to the conclusion that at least some micrococci—whether all the different species of micrococci, I do not know—constitute only a transitory form, and therefore have no right to be looked upon as the typical form of a genus.

Mr. President, inasmuch as you asked me to address this learned assembly totally unprepared, I hope the latter will treat with indulgence my rambling talk. I admit and explicitly state, some of the hints given, for as such I wish to have them considered,are almost entirely based upon clinical observation, and some of them are as yet insufficiently supported by such absolute proof as is required in scientific research. But if the same give an impulse, or point out the direction in which experiments and further research may lead to important results, I shall not regret that I yielded, unprepared as I am to your request.